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L4            14 DUP REM L3 (28 DUPLICATES REMOVED)
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MEDICONF, NUTRACEUT, PCTGEN, PHAR, PHARMAML, PROUSDDR, RDISCLOSURE, SYNTHLINE'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
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L4           14 DUP REM L3 (28 DUPLICATES REMOVED)
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ANSWER 15 OF 17 DGENE COPYRIGHT 2004 THOMSON DERWENT on STN
AN AAA12617 cDNA DGENE
TI New nucleic acids encode enzymes of wasp venom, are useful to treat insect sting allergy or immune system-related disorders and differ from the genomic sequences in that introns have been removed -
IN King T P
PA (UYRQ) UNIV ROCKEFELLER.
PI WO 2000018896 A1 20000406 72p
AI WO 1999-US23211 19991001
PRAI US 1998-166205 19981001
PSL Claim 9; Fig 4
DED 25 JUL 2000 (first entry)
DT Patent
LA English
OS 2000-293139 [25]
CR P-PSDB: AAY84614
DESC cDNA encoding a Pol a venom hyaluronidase polypeptide.
KW Pol a venom; hyaluronidase; paper wasp; immune response; immunogen; vespid venom; allergen-specific allergy; hymenoptera venom; autoimmune condition; allergic condition; viral infection; HIV; human immunodeficiency virus; Herpes Simplex virus; papilloma virus; ss.
ORGN Polistes annularis.
AB The present sequence encodes a Pol a venom **hyaluronidase** polypeptide, isolated from the paper wasp. The enzyme acts on the disaccharide unit of D-glucoonic acid and N-acetyl-D-glucosamine. The recombinant Polistinae venom is used to modulate an immune response to an immunogen in a mammal, particularly a vespid venom allergen-specific allergy, or allergy to other hymenoptera venom. Alternatively the venom enzyme is used to treat an immunologically affected disease or disorder, particularly a pathogenic disease or disorder, an autoimmune condition, an allergic condition, especially an allergy to hymenoptera venom, or a viral infection, especially human immunodeficiency virus (**HIV**), Herpes Simplex virus or papilloma virus. The enzyme is also useful to diagnose allergy.
NA 460 A; 213 C; 271 G; 329 T; 0 other
SQL 1273

d ibib abs l4 1-14

L4 ANSWER 1 OF 14 USPATFULL on STN
ACCESSION NUMBER: 2003:300795 USPATFULL
TITLE: Myeloid colony stimulating factor and uses thereof
INVENTOR(S): Frost, Gregory I., Solana Beach, CA, UNITED STATES
Borgstrom, Per, La Jolla, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003212021	A1	20031113	
APPLICATION INFO.:	US 2002-182088	A1	20021126	(10)
	WO 2001-US2575		20010125	

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Lisa A Haile J D, Gray Cary Ware & Freidenrich, Suite 1100, 4365 Executive Drive, San Diego, CA, 92121-2133
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
LINE COUNT: 745
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The identification of the HYAL1 hyaluronidase enzyme as a human plasma-derived myeloid colony-stimulating factor (CSF), herein designated CSF5-hyaluronidase, its recombinant production and methods of use are described. This protein may be used for the treatment of myelosuppression as may occur after irradiation, chemotherapy or other diseases where an increase in leukocyte levels may be beneficial. For example, CSF5 may be used to enhance the immune response to viral infection or other diseases associated with immune suppression.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 2 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2004:33610 BIOSIS
DOCUMENT NUMBER: PREV200400031748
TITLE: ENDOPLASMIC RETICULUM STRESS IN MUCOSAL SMOOTH MUSCLE CELLS UPREGULATES HYALURONAN DEPOSITION AND LEUKOCYTE ADHESION.
AUTHOR(S): Majors, Alana K. [Reprint Author]; Austin, Richard C.; de Motte, Carol A. la; Pyeritz, Reed E.; Strong, Scott A.
CORPORATE SOURCE: Cleveland, OH, USA
SOURCE: Digestive Disease Week Abstracts and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. T1165. e-file. Meeting Info.: Digestive Disease 2003. FL, Orlando, USA. May 17-22, 2003. American Association for the Study of Liver Diseases; American Gastroenterological Association; American Society for Gastrointestinal Endoscopy; Society for Surgery of the Alimentary Tract.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Jan 2004
Last Updated on STN: 7 Jan 2004
AB Endoplasmic reticulum (ER) stress is associated with inflammation, but the relationship between ER stress and inflammatory diseases is not known. We have previously shown that the inflammation typical of Crohn's disease and ulcerative colitis is associated with enhanced deposition of hyaluronan (HA) in the extracellular matrix of the intestinal mucosa. Although mucosal smooth muscle cells (M-SMCs) are recognized to produce HA, the mechanisms responsible for this abnormal HA accumulation remain poorly understood. Therefore, we examined the capacity of ER stress to modulate HA deposition by M-SMCs in cultures derived from human colon surgical specimens. Visualization of hyaluronan with affinity histochemistry and fluorescent confocal microscopy demonstrated little accumulation of HA in untreated cultures. In contrast, M-SMC cultures treated with tunicamycin,

an agent that strongly induces ER stress by interfering with glycosylation, demonstrated a matrix rich in HA that was present in both coat and novel, cable-like structures. Thapsigargin and A23187, which induce ER stress by altering calcium homeostasis, also upregulated the deposition of HA. Likewise, dextran sulfate, another agent that induces ER stress, and promotes intestinal inflammation in vivo, dramatically induced HA deposition. Leukocyte adhesion assays employing radiolabeled U937 cells or **peripheral blood mononuclear** leukocytes, demonstrated minimal leukocyte binding to untreated M-SMCs, but significantly increased adhesion (15-fold) when ER function of the M-SMCs was initially perturbed. The bound leukocytes were released by digestion with **hyaluronidase**, suggesting HA-mediated adhesion. Fluorescence microscopy confirmed that the HA-containing cables served as attachment sites for the leukocytes. This data indicates a novel mechanism exists through which ER stress of M-SMCs induces leukocyte adhesion by enhancing the accumulation of a unique form of hyaluronan and suggests that ER stress may contribute to the pathogenesis of inflammatory bowel disease by altering the extracellular matrix and its capacity to interact with leukocytes..

L4 ANSWER 3 OF 14 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2002:850139 CAPLUS
DOCUMENT NUMBER: 137:333123
TITLE: Hyaluronidase for treating retroviral infections
INVENTOR(S): Gallina, Damian J.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 11 pp., Cont.-in-part of U. S. Ser. No. 305,801.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002164321	A1	20021107	US 2002-50655	20020116
PRIORITY APPLN. INFO.:			US 1998-82185P	P 19980417
			US 1999-305801	A2 19990422

AB The present invention relates to **hyaluronidase** enzyme methods, vaccines and compns. for treating or preventing pathogenic infections, such as HIV, in a patient. The invention also relates to methods, vaccines and compns. for providing immunity against HIV infection in a patient comprising treating the patient with HIV virus or **HIV infected cells** that have been treated with **hyaluronidase**.

L4 ANSWER 4 OF 14 USPATFULL on STN
ACCESSION NUMBER: 2002:148584 USPATFULL
TITLE: HUMAN T CELL CLONE SPECIFIC FOR RHEUMATOID ARTHRITIS
INVENTOR(S): TOYOSAKI-MAEDA, TOMOKO, HYOGO, JAPAN
SUZUKI, RYUJI, NARA, JAPAN
TSURUTA, YUJI, OSAKA, JAPAN
TAKEMOTO, HIROSHI, HYOGO, JAPAN

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002076725	A1	20020620	
APPLICATION INFO.:	US 1998-142755	A1	19980914	(9)
	WO 1997-JP774		19970312	

	NUMBER	DATE
PRIORITY INFORMATION:	JP 1996-56022	19960313

WO 1996-JP3082 19961023
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FOLEY & LARDNER, WASHINGTON HARBOUR, 3000 K STREET NW SUITE 500, PO BOX 25696, WASHINGTON, DC, 20007-8696
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 865
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A human T cell clone recognizing an antigen expressed by a synovial cell of a rheumatoid arthritis (RA) patient in HLA-DR-restricted manner is disclosed, which clone is very useful in exploring the pathogenesis of RA and developing a method for treating and preventing RA.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 5 OF 14 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 2
ACCESSION NUMBER: 2002436991 EMBASE
TITLE: Glucocorticoids enhance interleukin-4 production to neo-antigen (hyaluronidase) in children immunocompromised with cytostatic drugs.
AUTHOR: Edelbauer M.; Gerstmayr M.; Loibichler C.; Jost E.; Huemer M.; Urbanek R.; Szepfalusi Z.
CORPORATE SOURCE: Z. Szepfalusi, Department of Paediatrics, AKH, Wahringergurtel 18-20, A-1090 Wien, Austria. ZSOLT.SZEPFALUSI@akh-wien.ac.at
SOURCE: Pediatric Allergy and Immunology, (2002) 13/5 (375-380). Refs: 23
ISSN: 0905-6157 CODEN: PALUEE
COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Immunoglobulin E (IgE)-mediated immediate-type allergic reactions to **hyaluronidase** have been observed in children with central nervous system (CNS) tumors. Glucocorticoids, used as therapy for brain edema, are discussed controversially as T helper 2 (Th2) stimulatory factors. In this study we investigated the role of glucocorticoids on a Th2 cytokine-promoting effect in children with CNS tumors. **Peripheral blood mononuclear** cells (PBMCs) from: 29 children suffering from malignant brain tumors, of whom 23 received short-term glucocorticoid treatment (for 3-4 days) during the course of chemotherapy; 18 children with nephrotic syndrome or renal transplantation receiving long-term glucocorticoid treatment; and 13 healthy children, were incubated with phytohemagglutinin (PHA) and/or anti-CD28 monoclonal antibody (mAb) and, in a second approach, with **hyaluronidase**. The concentrations of Th cell-mediated cytokines - interleukin (IL)-4, IL-10, and interferon-γ (IFN-γ)-were measured in supernatants. The IL-4 production of PBMCs incubated with PA/anti-CD28 mAb from children with repeated coadministration of glucocorticoids, **hyaluronidase** , and cytostatic drugs (median: 249.9 pg/ml; range: 234.4-261.7) was significantly higher ($p < 0.0001$) than IL-4 production of PBMC from children of all the other groups (median: 86.18; range: 16.0-212.5). There was no significant difference in the levels of IL-10 and IFN-γ within the groups. PBMCs stimulated only with **hyaluronidase** failed to produce detectable levels of cytokines. The results of this study indicate that repeated co-administration of glucocorticoids and **hyaluronidase** (a neo-antigen) enhance IL-4 production in vitro and

thus may induce the production of specific IgE antibodies in children immunocompromised with cytostatic drugs. **Hyaluronidase** itself does not stimulate in vitro IL-4 synthesis in PBMCs of children receiving cytostatic drugs. .COPYRGT. 2002 Blackwell Munksgaard.

L4 ANSWER 6 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 3
ACCESSION NUMBER: 2002:612982 BIOSIS
DOCUMENT NUMBER: PREV200200612982
TITLE: Human monocytes synthesize hyaluronidase.
AUTHOR(S): Girard, Nicole [Reprint author]; Maingonnat, Catherine; Bertrand, Philippe; Tilly, Herve; Vannier, Jean-Pierre; Delpech, Bertrand
CORPORATE SOURCE: Laboratory of Molecular Oncology, Centre Henri-Becquerel, F76000, Rouen, France
ngirard@rouen.fnclcc.fr
SOURCE: British Journal of Haematology, (October, 2002) Vol. 119, No. 1, pp. 199-203. print.
CODEN: BJHEAL. ISSN: 0007-1048.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Nov 2002
Last Updated on STN: 27 Nov 2002
AB The involvement of hyaluronic acid (HA) oligosaccharides and blood-derived mononuclear cells in inflammatory processes prompted us to determine whether **peripheral blood mononuclear** cells (PBMC) possess **hyaluronidase** activity. PBMC were incubated with macromolecular-tritiated HA at pH 3.8 and supernatants were analysed by size exclusion chromatography to reveal digestion of HA. This digestion was due to the CD14-positive (CD14+), adherent, non-specific esterase-positive, subpopulation of PBMC. **Hyaluronidase** activity (72 kDa) was found in aqueous and non-ionic detergent PBMC extracts but not in the medium in which the cells had been cultured. These results indicate that **hyaluronidase** is, at least in part, linked to the membrane rather than excreted. Hence, monocytes have one or more hyaluronidases that can generate a pool of active HA fragments within tissues. **Hyaluronidase** activity was also found in 3/3 myelomonocytic lineage leukaemias but not in 3/3 lymphoblastic leukaemias.

L4 ANSWER 7 OF 14 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
ACCESSION NUMBER: 2002-06955 BIOTECHDS
TITLE: New bee venom polypeptides, useful for modulating immune responses e.g. in individual hypersensitive to the venom and for identifying individual at risk for bee venom hypersensitivity;
recombinant glycosylated protein production and hybridoma-derived monoclonal antibody for use in recombinant vaccine
AUTHOR: SPERTINI F
PATENT ASSIGNEE: ECOLE POLYTECHNIQUE FEDERALE LAUSANNE
PATENT INFO: WO 2001088085 22 Nov 2001
APPLICATION INFO: WO 2000-IB1736 18 Feb 2000
PRIORITY INFO: US 2000-506978 18 Feb 2000
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2002-082988 [11]
AN 2002-06955 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - A substantially pure polypeptide (I) derived from bee venom Api m 6, comprising an amino acid (aa) sequence at least 70% identical to a fully defined sequence (S1) of 67 aa as given in the specification, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following: (1) an antibody (Ab) which binds to (I); (2) a hybridoma (II)

producing an antibody which binds to the same epitope to which the monoclonal antibody produced by the 5E11 (Accession number undefined) binds; (3) a composition (III) comprising polypeptide fragments of 6-72 amino acids of the Api m 6 protein; (4) a pharmaceutical composition (IV) comprising (I) and a carrier; and (5) a kit comprising in one or more containers, a substance selected from (I), a overlapping polypeptide fragments of (I) and Ab.

WIDER DISCLOSURE - The following are disclosed: (1) variant of (I); (2) fragments of (I); (3) modified forms of (I), its fragments and their variants; and (4) chimeric or fusion proteins of (I).

BIOTECHNOLOGY - Preparation: (I) is produced by standard DNA recombinant techniques. Preferred Polypeptide: (I) preferably comprises an aa sequence 90 % identical to (S1); or a sequence at least 70% identical to a 69, 71 or 73 aa sequence as given in the specification. (I) is preferably glycosylated, binds to a human immunoglobulin E (IgE) antibody, stimulates T-cell proliferation, and binds to the monoclonal antibody 5E11. Preferred Antibody: Ab is preferably monoclonal, polyclonal or humanized antibody, where the monoclonal antibody binds to the same epitope to which the monoclonal antibody produced by hybridoma 5E11 binds. Preferred Hybridoma: (II) is preferably hybridoma 5E11. Preferred Composition: (III) comprising fragments of (I), preferably comprises fragments of 20-100, more preferably 40-60 amino acids. In (III), at least one polypeptide has an amino acid sequence that overlaps by at least 3 amino acids preferably 5-10 amino acids, with at least one other polypeptide in the composition. (III) comprises a set of polypeptide fragments that map the total length of (I). (IV) further comprises a second bee venom polypeptide, selected from phospholipase A2, **hyaluronidase**, allergen C, mellitin, adolapin, minimine, acid phosphatase, protease inhibitor, and glycosylated IgE-binding proteins, or their analogs or derivatives.

ACTIVITY - Immunosuppressant. No supporting data is given.

MECHANISM OF ACTION - Vaccine.

USE - (I) is useful for modulating an immune response, preferably to inhibit an immune reaction by the subject against (I). (I) is useful for identifying an individual at risk for bee venom hypersensitivity. The method comprises administering (I) to the individual and measuring an immune response raised against (I), where a detectable immune response indicates that the individual is at risk for bee venom hypersensitivity and where (I) is administered intradermally at a dosage of less than 1 microgram/ml. Ab is useful for purifying (I). The method comprises contacting (I) with Ab to form (I)-Ab complex, isolating the complex formed and recovering (I) from the complex (all claimed).

ADMINISTRATION - (I) is administered through parenteral e.g. intravenous, intradermal, subcutaneous, oral (e.g. inhalation), transdermal (topical), transmucosal or rectal routes. No specific dosage detail is given.

EXAMPLE - The Api m 6 bee venom protein was identified in studies examining the reactivity of immunoglobulin (Ig)E-sera derived from patients hypersensitive to purified bee venom (BV) proteins. Serum and **peripheral blood mononuclear** cells (PBMC)
were obtained from BV hypersensitive patients (grade II-IV, according to Mueller's classification) Mueller, J Asthma Res 3:331-333 (1966). All patients had BV specific IgE (at least 0.35 kU/I) and positive intradermal skin tests (at least 0.1 microgram/ml). BV proteins were separated by 15% SDS-PAGE (sodium dodecyl sulfate-polyacrylamide gel electrophoresis) under non-reducing conditions and blotted to PVDF membranes in CAPS/methanol buffer (10 mM CAPS, 10% methanol, pH 11). Membranes were blocked with non-fat milk (5%) in phosphate buffered salt (PBS) solution containing 0.1% Tween 20 (PBS-Tween), then incubated with patient's sera (1/10 in PBS-Tween) for 24 hours at 4 degrees C. Specific IgE binding was detected using a biotinylated monoclonal mouse anti-human IgE antibody (pharmingen, Hamburg, Germany) followed by incubation with streptavidin conjugated horseradish peroxidase (HRP) (UBI, Lucerna Chem AG, Luzern, Switzerland). Peroxidase reactivity was visualized by

enhanced chemiluminescence (ECL, Amersham, UK). Analysis of IgE sera from 43 patients reactive with separated BV proteins revealed a previously undescribed band at about 8 kD in 18 (42%) of the samples. The 8 kDa protein corresponding to the observed 8 kDa band was purified from other BV proteins by size exclusion chromatography. Chromatography was performed by lyophilizing whose BV (Apis mellifera) (Latoxan, Rosans, France) in 50% formic acid. Particles were removed by centrifugation and filtration prior to sample application to BioRad P-60 column (2.5x100 cm) (BioRad, Glattbrugg, Switzerland) equilibrated in 50% formic acid. Acidic conditions were used to minimize melittin tetramer formation. Bello, et al., Biochemistry 21:461-465 (1982). Fractions of 4 ml were collected at a flow rate of 6.5 ml/h. Each fraction was lyophilized, dissolved in 0.02 N acetic acid and analyzed by SDS-PAGE. Laemmili, Nature 227:680-685 (1970. Fractions containing the 8 kDa band eluted in a broad peak between the peaks of two other bee venom proteins, PLA2 and melittin. Matrix assisted laser ionization-time of flight (MALDI-TOF) mass spectrometry analysis of these fractions revealed the presence of four proteins with molecular weights of 7,190, 7400, 7,598 and 7,808 Da. The four proteins were further purified by reverse phase high performance liquid chromatography (HPLC) using two runs through a C4 column (Phenomex W-Porex; 250x46 mm; Rancho Palos Verdes, CA, USA). Water acetonitrile gradient was used for separation (buffer A:10% acetonitrile, 0.1% trifluoroacetic acid in water; buffer B:90% acetonitrile, 0.1% trifluoroacetic acid in water). All four proteins were recognized by IgE from a BV hypersensitive patient that was positive for the 8 kDa protein in the initial screening, were named Api m 6.01, Api m 6.02, and Api m 6.03, and Api m 6.04, respectively. (32 pages)

L4 ANSWER 8 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 4

ACCESSION NUMBER: 2000:430609 BIOSIS
DOCUMENT NUMBER: PREV200000430609
TITLE: Mononuclear leukocytes preferentially bind via CD44 to hyaluronan on human intestinal mucosal smooth muscle cells after virus infection or treatment with poly(IcntdotC).
AUTHOR(S): de la Motte, Carol A.; Hascall, Vincent C.; Calabro, Anthony; Yen-Lieberman, Belinda; Strong, Scott A. [Reprint author]
CORPORATE SOURCE: Lerner Research Inst., Cleveland Clinic Foundation, 9500 Euclid Ave., NB3, Cleveland, OH, 44195, USA
SOURCE: Journal of Biological Chemistry, (Oct. 22, 1999) Vol. 274, No. 43, pp. 30747-30755. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Oct 2000
Last Updated on STN: 10 Jan 2002

AB Pathological changes in inflammatory bowel disease include an increase in intestinal mucosal mononuclear leukocytes and hyperplasia of the muscularis mucosae smooth muscle cells (M-SMCs). Because virus infections have correlated with disease flare, we tested the response of cultured M-SMCs to respiratory syncytial virus, measles virus, and the viral analogue, poly(IcntdotC). Adhesion of U937 cells and **peripheral blood mononuclear** cells was used to measure the leukocyte-interactive potential of M-SMCs. Untreated M-SMCs, only minimally adhesive for leukocytes, bound U937 cells after treatment with respiratory syncytial virus or measles virus. Mononuclear leukocytes also bound to poly(IcntdotC)-treated M-SMCs. Although both vascular cell adhesion molecule-1 mRNA and protein increased 3-4-fold in poly(IcntdotC)-treated M-SMC cultures, U937 cell adhesion was not blocked by an anti-vascular cell adhesion molecule-1 monoclonal antibody. However, **hyaluronidase** digestion of poly(IcntdotC)- or virus-treated M-SMCs dramatically reduced leukocyte adhesion (apprx75%). Fluorophore-assisted carbohydrate electrophoresis demonstrated a

apprx3-fold increase in surface-bound hyaluronan on poly(IcntdotC)-treated M-SMCs compared with untreated controls. In addition, pretreatment of mononuclear cells with a blocking anti-CD44 antibody, greatly decreased adhesion to poly(IcntdotC)-treated M-SMCs. Recognition of this virus-induced hyaluronan/CD44 mechanism of mesenchymal cell/leukocyte interaction introduces a new avenue in the research of gut inflammation.

L4 ANSWER 9 OF 14 DISSABS COPYRIGHT (C) 2004 ProQuest Information and Learning Company; All Rights Reserved on STN

ACCESSION NUMBER: 96:35359 DISSABS Order Number: AAI9617526
TITLE: THE ROLE OF CD44 IN HIV INFECTION (MONOCYTES, ADHESION, IMMUNE DEFICIENCY, HYALURONIC ACID)
AUTHOR: GUO, MARGARET MING-TI [PH.D.]
CORPORATE SOURCE: THE JOHNS HOPKINS UNIVERSITY (0098)
SOURCE: Dissertation Abstracts International, (1996) Vol. 57, No. 1B, p. 225. Order No.: AAI9617526. 198 pages.
DOCUMENT TYPE: Dissertation
FILE SEGMENT: DAI
LANGUAGE: English
ENTRY DATE: Entered STN: 19960708
Last Updated on STN: 19960708

AB We have found that HIV-1 infection of two unrelated monocytic cell lines (THP-1 and MonoMac) results in a new homotypic adhesion phenotype. Whereas uninfected cells grow as single cell suspensions, **HIV-infected cells** grow as large aggregates. When the expression of adhesion molecules was investigated, CD44 was almost completely depleted from the surface of **HIV-infected cells**. This project aims to define the mechanism of CD44 loss and to investigate the potential role of CD44 as an accessory molecule in HIV infection. Immunoprecipitation, western blot analysis, and ELISA assays showed that CD44 was not found on the surface, in internal complexes, or in the culture supernatant. Northern blot analysis showed similar RNA patterns in **HIV-infected cells** and uninfected control cells in both size and quantity. Pulse chase experiments showed that CD44 core protein could not be detected in the infected cells. Thus the loss of CD44 was most likely due to a translational block. Attempts to restore CD44 expression with expression vectors were not successful. CD44 loss in monocytes infected in vitro and from HIV-1 infected patients could not be demonstrated. To evaluate CD44 as a potential co-receptor for HIV, antibody blocking experiments and infection of a CD44 negative cell line were carried out. An anti-CD44 mAb partially inhibited HIV-1 infection of a monocytic cell line in a dose dependent fashion. A CD44-negative mutant cell line was established from a monocytic cell line. The mutant and parental cell lines showed similar susceptibility to HIV-1 infection and cytopathic effects. Therefore, CD44 may play an accessory role in HIV-1 infection but not a necessary role. Finally, the functional significance of the CD44 loss was investigated. Both the uninfected and infected monocytic cells showed no binding to hyaluronic acid even after stimulation with phorbol ester or treatment with **hyaluronidase**. However, in another cell line that was inducible by phorbol ester to bind hyaluronate, the virus bound to hyaluronate only when it was produced in phorbol ester-stimulated cells. Therefore, the presence of CD44 on viral surface may have functional significance.

L4 ANSWER 10 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 5

ACCESSION NUMBER: 1994:437023 BIOSIS
DOCUMENT NUMBER: PREV199497450023
TITLE: Antigen-presenting capacity in normal human dermis is mainly subserved by CD1a+ cells.
AUTHOR(S): Sepulveda-Merrill, C.; Mayall, S.; Hamblin, A. S.; Breathnach, S. M. [Reprint author]
CORPORATE SOURCE: St. John's Inst. Dermatol., St. Thomas' Hosp., Lambeth

Palace Road, London SE1 7EH, UK
SOURCE: British Journal of Dermatology, (1994) Vol. 131, No. 1, pp. 15-22.
CODEN: BJDEAZ. ISSN: 0007-0963.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Oct 1994
Last Updated on STN: 11 Oct 1994

AB A proposed role for antigen-presenting dermal dendrocytes in the pathogenesis of many dermal inflammatory skin diseases remains speculative. We therefore sought to determine the phenotype and functional characteristics of antigen-presenting cells isolated from normal human dermis. Normal adult human skin was incubated overnight with dispase at 4 degree C, the epidermis was removed, and the residual dermal preparation was then minced and digested with a mixture of **hyaluronidase**, collagenase, and DNAase at 37 degree C, prior to filtration through mesh. Dermal cell suspensions thus obtained were stained using specific monoclonal antibodies, and analysed by fluorescence microscopy or flow cytometry. Mean values were as follows: CD45+ leucocytes 39%, HLA-DR+ cells 39%, Ulex europaeus agglutinin I+ endothelial cells 26%, CD1a+ cells 3.9%, CD11b+ cells 16%, CD11c+ cells 6%. Mitomycin C-treated crude dermal cell suspensions induced allostimulation of **peripheral blood mononuclear** cells in a 7-day culture, as assessed by 3H-TdR incorporation. Depletion of CD1a+ Langerhans-like cells from the dermal cell preparation, by 95, 74 and 90% in three separate experiments using immunomagnetic beads, reduced 3H-TdR incorporation at optimal responder-to-stimulator cell ratios by 90, 64, and 87%, respectively. Our findings suggest that, in normal human dermis, the great majority of the alloantigen-presenting capacity resides in the CD1a+ Langerhans cell-like dendritic antigen-presenting cell population, and not to any great extent in either CD1a- macrophage-like cells, or HLA-DR+ endothelial cells. The relationship of the CD1a+ dermal antigen-presenting cells to the Langerhans cell lineage remains to be determined.

L4 ANSWER 11 OF 14 DRUGU COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 1994-09849 DRUGU S
TITLE: Synovial Tissue Implants from Patients with Rheumatoid Arthritis Cause Cartilage Destruction in Knee Joints of SCID.bg Mice.
AUTHOR: Sack U; Kuhn H; Ergmann J; Kinne R W; Vogt S; Jungmichel D
CORPORATE SOURCE: Univ.Leipzig; Univ.Erlangen-Nuremberg
LOCATION: Bad Duben, Germany,West
SOURCE: J.Rheumatol. (21, No. 1, 10-16, 1994) 4 Fig. 21 Ref.
CODEN: JRHUA9 ISSN: 0315-162X
AVAIL. OF DOC.: Institut fuer Klinishe Immunologie der Universitaet Leipzig, Technikum Analytikum, Linnestrasse 3, 04103 Leipzig, Germany. (7 authors).
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature
AN 1994-09849 DRUGU S

AB A mouse model of joint destruction initiated by human inflammatory cells from patients with rheumatoid arthritis (RA) was established where synovial membrane implants induced pannus formation and erosion of cartilage. Implantation of normal synovial membrane and control tissues like human thymus produced only a mild and transient synovitis. This method was not successful with human **peripheral blood mononuclear** cells (PMNC), T-cell lines reactive to mouse or rat collagen type II and synovial mononuclear cells because of their immigration from the knee joint without causing destruction. Cell immigration was reduced but not prevented by pre-activation with mitogens. This model is useful for studying pathogenetic aspects of

joint destruction as well as effects of new drugs or novel treatment strategies.

ABEX Methods SCID.bg mice had single cell suspensions injected into knee joints or tissue grafted into the joints. Results When PMNC, collagen-reactive T-cell lines and synovial membrane cell (SMNC) suspensions left the joint space and migrated into the mouse synovial membrane reaching peak numbers 12 hr after injection into the knee joint. Preactivation of PMNC or SMNC by a strong polyclonal activator reduced migration. Simultaneous injection of mouse or rat collagen or actionized mouse collagen or pretreatment of the joint with **hyaluronidase** did not induced formation of an inflammatory focus or persistence of human cells for more than a few hrs. No erosions were seen in adjacent cartilage. When inflammed synovial membrane from RA patients was implanted into the knee joints, it induced cartilage and bone erosion similar to that induced by pannus tissue. Control experiments of surgery with no transfer of tissue caused no destruction and transfer of human thymus tissue and normal synovial membrane induced only a mild, transient synovitis. 7 Days after implantation at the site of cartilage destruction there were a large number of human macrophages but human T cells were very scant. Human pannus tissue rich in CD68+ macrophages eroded mouse bone; mouse granulocytes accumulated close to or inside the human tissue. (K65/JC)

L4 ANSWER 12 OF 14 JICST-EPlus COPYRIGHT 2004 JST on STN

ACCESSION NUMBER: 880318076 JICST-EPlus

TITLE: Ferritin contents in leukocytes from patients with rhematoid arthritis.

AUTHOR: NISHIYA KOJI; SHIRAKAMI TOSHIAKI; HATANO MAKOTO; YAMAMURA MASAHIRO; KAWABATA FUKIKO; YOSHINAGA YASUHIKO; HIRAKI YOSHIO; AONO KANAME
EZAWA HIDEMITSU

CORPORATE SOURCE: Okayama Univ., School of Medicine
Kurashiki Kosai Hospital

SOURCE: Ensho (Japanese Journal of Inflammation), (1987) vol. 7, no. 6, pp. 541-545. Journal Code: Y0899A (Fig. 2, Tbl. 3, Ref. 18)
CODEN: ENSHEE; ISSN: 0389-4290

PUB. COUNTRY: Japan

DOCUMENT TYPE: Journal; Article

LANGUAGE: Japanese

STATUS: New

AB Ferritin contents in peripheral blood or synovial fluid leukocytes from 24 RA patients and 14 healthy donors were measured by 2-sites immunoradiometric assay as supernatants after cells with 5 times repetition of freezing and thawing were centrifuged at 3000 r.p.m. for 20min. Ferritin contents per a single cell(fg/cell) were calculated from the value of measured supernatants divided by the number of cells. Polymorphonuclear cells(PMN) and mononuclear cells(MNC) were separated from buffy coats or synovial fluid with heparin(10u/ml) and **hyaluronidase**(20u/ml) by Conray-Ficoll gradient sedimentation method. **Peripheral blood mononuclear** cells(1*106cells/ml; PBM) were suspended in RPMI 1640 medium containing 10% fetal calf serum and cultured in 5% CO2 incubator at 37.DEG.C for 7 days with or without addition of different concentration of ferric citrate ranged from 0.01 to 1mM. Ferritin contents in PBM were increased with addition of ferric citrate in dose-dependent manner. Ferritin contents in peripheral blood PMN(mean±SEM=5.3±2.6fg/cell, n=16) and MNC(9.3±3.3fg/cell, n=16) of RA patients were not significantly different from that of healthy controls(PMN: 4.1±2.6fg/cell, n=14 and MNC: 11.1±2.7fg/cell, n=15). It was, however, found in both groups that ferritin contents in MNC were higher than that in PMN. Ferritin contents in synovial fluid PMN(29.7±9.5fg/cell, n=16) and MNC(62.4.+-.7.1fg/cell, n=16) of RA patients were remarkably higher than that in peripheral blood leukocytes.(author abst.)

L4 ANSWER 13 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 6
ACCESSION NUMBER: 1986:240954 BIOSIS
DOCUMENT NUMBER: PREV198682005458; BA82:5458
TITLE: ULTRASTRUCTURAL FEATURES OF THE LYMPHOCYTE-STIMULATED HALOS PRODUCED BY HUMAN GLIOMA-DERIVED CELLS IN-VITRO.
AUTHOR(S): OBERC-GREENWOOD M A [Reprint author]; MUUL L M; GATELY M K; KORNBLITH P L; SMITH B H
CORPORATE SOURCE: SURGICAL NEUROL BRANCH, NINCDS, BUILD 10A, ROOM 3E68, BETHESDA, MD 20205, USA
SOURCE: Journal of Neuro-Oncology, (1986) Vol. 3, No. 4, pp. 387-396.
CODEN: JNODD2. ISSN: 0167-594X.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 7 Jun 1986
Last Updated on STN: 7 Jun 1986

AB Many glioma-derived cell lines have the capability of escaping cell-mediated immune attack. One mechanism of escape is the secretion of a **hyaluronidase**-sensitive mucopolysaccharide coat by these cells. This coat prevents contact and tumor cell killing by specific cytolytic allogeneic lymphocytes. The production of the coat by the tumor cells in stimulated by a macromolecular factor released by **peripheral blood mononuclear** (PBMC) cells in culture. We have examined the morphologic and ultrastructural features of this extracellular matrix. Three coat-producing lines were studied. Under phase contrast light microscopy, the coat is a clear pericellular 'halo'. To stain this zone, ruthenium red and Alcian Blue 8 G stains, which bind to acid mucopolysaccharides (to a large extent, hyaluronic acid), were used. The two stains produced similar results. With light microscopy, a weblike pattern of stain was evident throughout the halo region. With transmission electron microscopy, staining was found along the plasma membrane of the glioma cells and their microvilli, stretching in long, branching filaments from these surfaces and, in some instances, from one microvillus to the next. Since mucopolysaccharide matrices have a large aqueous component, it was necessary to determine whether dehydration alters the stain pattern. Therefore, undehydrated ruthenium red stained specimens from each culture were embedded in Quetal 651 (Ted Pella, Inc., Tustin, CA), a water soluble plastic. No morphologic differences were noted between the hydrated and dehydrated specimens. This study indicates that numerous long microvilli and a secreted mucopolysaccharide matrix are important structural elements of the lymphocyte-stimulated tumor cell halo in vitro. The mechanism by which the PBMC factor stimulates coat formation and the importance of the coat in in vivo tumor defenses remain to be elucidated.

L4 ANSWER 14 OF 14 KOSMET COPYRIGHT 2004 IFSCC on STN
ACCESSION NUMBER: 11302 KOSMET
FILE SEGMENT: scientific, technical
TITLE: ANTIGEN-PRESENTING CAPACITY IN NORMAL HUMAN DERMIS IS MAINLY SUBSERVED BY CD1A+ CELLS
AUTHOR: SEPULVEDA MERRILL C (ST JOHN'S INSTITUTE OF DERMATOLOGY, ST THOMAS' HOSPITAL, LONDON UK); MAYALL S; HAMBLIN A S; BREATHNACH S M
SOURCE: BRIT J DERMATOL, 1994, 131(1), 15-22, 44 REFS
DOCUMENT TYPE: Journal
LANGUAGE: English

AN 11302 KOSMET FS scientific, technical
AB A proposed role for antigen-presenting dermal dendrocytes in the pathogenesis of many dermal inflammatory skin diseases remains speculative. We therefore sought to determine the phenotype and functional characteristics of antigen-presenting cells isolated from

normal human dermis. Normal adult human skin was incubated overnight with dispase at 4 degrees, the epidermis was removed, and the residual dermal preparation was then minced and digested with a mixture of **hyaluronidase**, collagenase, and DNAase at 37 degrees, prior to filtration through mesh. Dermal cell suspensions thus obtained were stained using specific monoclonal antibodies, and analysed by fluorescence microscopy or flow cytometry. Mean values were as follows: CD45+ leucocytes 39%, HLA-DR + cells 39%. Ulex europaeus agglutin I+ endothelial cells 26%, CD1a+ cells 3.9%, CD11b+ cells 16%, CD11c+ cells 6%. Mitomycin C-treated crude dermal cell suspensions induced allostimulation of **peripheral blood mononuclear** cells in a 7-day culture, as assessed by 3H-TdR incorporation. Depletion of CD1a+ Langerhans-like cells from the dermal cell preparation, by 95,74 and 90% in three separate experiments using immunomagnetic beads, reduced 3H-TdR incorporation at optimal responder-to-stimulator cell ratios by 90, 64, and 87%, respectively. Our findings suggest that, in normal human dermis, the great majority of the alloantigen-presenting capacity resides in the CD1a+ Langerhans cell-like dendritic antigen-presenting cell population, and not to any great extent in either CD1a- macrophage-like cells, or HLA-DR+ endothelial cells. The relationship of the CD1a+ dermal antigen-presenting cells to the Langerhans cell lineage remains to be determined

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ANSWER 9 OF 17 DISSABS COPYRIGHT (C) 2004 ProQuest Information and Learning Company; All Rights Reserved on STN

AN 96:35359 DISSABS Order Number: AAI9617526

TI THE ROLE OF CD44 IN HIV INFECTION (MONOCYTES, ADHESION, IMMUNE DEFICIENCY, HYALURONIC ACID)

AU GUO, MARGARET MING-TI [PH.D.]

CS THE JOHNS HOPKINS UNIVERSITY (0098)

SO Dissertation Abstracts International, (1996) Vol. 57, No. 1B, p. 225. Order No.: AAI9617526. 198 pages.

DT Dissertation

FS DAI

LA English

ED Entered STN: 19960708
Last Updated on STN: 19960708

AB We have found that **HIV**-1 infection of two unrelated monocytic cell lines (THP-1 and MonoMac) results in a new homotypic adhesion phenotype. Whereas uninfected cells grow as single cell suspensions, **HIV**-infected cells grow as large aggregates. When the expression of adhesion molecules was investigated, CD44 was almost completely depleted from the surface of **HIV**-infected cells. This project aims to define the mechanism of CD44 loss and to investigate the potential role of CD44 as an accessory molecule in **HIV** infection. Immunoprecipitation, western blot analysis, and ELISA assays showed that CD44 was not found on the surface, in internal complexes, or in the culture supernatant. Northern blot analysis showed similar RNA patterns in **HIV**-infected cells and uninfected control cells in both size and quantity. Pulse chase experiments showed that CD44 core protein could not be detected in the infected cells. Thus the loss of CD44 was most likely due to a translational block. Attempts to restore CD44 expression with expression vectors were not successful. CD44 loss in monocytes infected in vitro and from **HIV**-1 infected patients could not be demonstrated. To evaluate CD44 as a potential co-receptor for **HIV**, antibody blocking experiments and infection of a CD44 negative cell line were carried out. An anti-CD44 mAb partially inhibited **HIV**-1 infection of a monocytic cell line in a dose dependent fashion. A CD44-negative mutant cell line was established from a monocytic cell line. The mutant and parental cell lines showed similar susceptibility to **HIV**-1 infection and cytopathic effects. Therefore, CD44 may play an accessory role in **HIV**-1 infection but not a necessary role. Finally, the functional significance of the CD44 loss was investigated. Both the uninfected and infected monocytic cells showed no binding to hyaluronic acid even after stimulation with phorbol ester or treatment with **hyaluronidase**. However, in another cell line that was inducible by phorbol ester to bind hyaluronate, the virus bound to hyaluronate only when it was produced in phorbol ester-stimulated cells. Therefore, the presence of CD44 on viral surface may have functional significance.

CC 0982 HEALTH SCIENCES, IMMUNOLOGY; 0307 BIOLOGY, MOLECULAR; 0379 BIOLOGY, CELL